AMENDMENTS TO THE CLAIMS

Please amend claims 54, 60, 68-71, 76, 77, 79-81, 83-84, 86-87, 89-90. Please add new claims 91-113.

1-53. (Canceled)

54. (Currently Amended) A method for inhibiting lymphotoxin-β-receptor (LT-β-R) signaling in a subject having a Th1 cell-mediated autoimmune disorder or a Th1 cell-mediated chronic inflammatory disease comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT-β-R blocking agent, and a pharmaceutically acceptable carrier, wherein the LT-β-R blocking agent comprises a soluble LT-β-R or an antibody directed against LT-β-R.

55-56. (Canceled)

- 57. (Previously Presented) The method according to claim 54, wherein the subject is a human.
- 58. (Previously Presented) The method according to claim 54, wherein the LT- β -R blocking agent comprises a soluble LT- β -R having a ligand binding domain that can selectively bind to a surface LT ligand.
- 59. (Previously Presented) The method according to claim 54, wherein the LT- β -R blocking agent comprises a soluble LT- β -R fused to one or more heterologous protein domains.
- 60. (Currently Amended) The method according to claim $\underline{111}$ 54, wherein the LT- β -R blocking agent comprises a monoclonal antibody directed against LT- β -R.

61-65. (Canceled)

66. (Previously Presented) The method according to claim 58, wherein the soluble LT- β -R is administered in an amount sufficient to coat LT- β ligand -positive cells for 1 to 14 days.

67. (Canceled)

- 68. (Currently Amended) The method according to claim <u>59</u> 58, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.
- 69. (Currently Amended) The method according to claim <u>59</u> 58, wherein the pharmaceutical composition is administered to the subject via oral administration or parenteral administration.
- 70. (Currently Amended) The method according to claim <u>59</u> 58, wherein the pharmaceutical composition is administered <u>to the subject</u> via parenteral administration selected from the group consisting of subcutaneous administration, intravenous administration, and intralesional administration.
- 71. (Currently Amended) A method for inhibiting lymphotoxin-β receptor (LT-β-R) signaling in a subject having a Th1 cell-mediated autoimmune disorder or a Th1 cell-mediated ehronic inflammatory disorder comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT-β-R blocking agent, and a pharmaceutically acceptable carrier, wherein the LT-β-R blocking agent comprises a soluble LT-β-R fused to one or more heterologous protein domains.
- 72. (Previously Presented) The method according to claim 71, wherein the heterologous protein domain comprises a human immunoglobulin Fc domain.
- 73. (Previously Presented) The method according to claim 71, wherein the soluble LT- β -R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT- β -R ligand binding domain.

74. (Previously Presented) The method according to claim 73, wherein the heterologous domain further comprises a human immunoglobulin Fc domain.

- 75. (Previously Presented) The method according to claim 74, wherein the composition is administered to the subject at a dose of about 1 mg/kg.
- 76. (Currently Amended) The method according to claim 74, wherein the composition is administered to the subject via oral administration or parenteral administration.
- 77. (Currently Amended) The method according to claim 74, wherein the composition is administered to the subject via parenteral administration selected from the group consisting of subcutaneous administration, intravenous administration, and intralesional administration.

78. (Canceled)

- 79. (Currently Amended) The method according to claim 71, wherein the autoimmune disorder is selected from the group consisting of psoriasis, rheumatoid arthritis, diabetes mellitus, multiple sclerosis, sympathetic ophthalmia, and uveitis.
- 80. (Currently Amended) The method according to claim 100 71, wherein the chronic inflammatory disorder is selected from the group consisting of inflammatory bowel disease, Crohn's disease, and ulcerative colitis.
- 81. (Currently Amended) A method for inhibiting lymphotoxin-β receptor (LT-β-R) signaling in a subject having a Th1 cell-mediated autoimmune disorder or a Th1 cell-mediated ehronic inflammatory disorder comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT-β-R blocking agent comprising a soluble LT-β-R fused to a heterologous domain comprising a human immunoglobulin Fc domain, and a pharmaceutically acceptable carrier, wherein the soluble LT-β-R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT-β-R ligand binding domain.

82. (Previously Presented) The method according to claim 81, wherein the composition is administered to the subject at a dose of about 1 mg/kg.

- 83. (Currently Amended) The method according to claim 81, wherein the composition is administered to the subject via oral administration or parenteral administration.
- 84. (Currently Amended) The method according to claim 81, wherein the composition is administered via parenteral administration selected from the group consisting of subcutaneous administration, intravenous administration, and intralesional administration.

85. (Canceled)

- 86. (Currently Amended) The method according to claim 81, wherein the autoimmune disorder is selected from the group consisting of psoriasis, rheumatoid arthritis, diabetes mellitus, multiple sclerosis, sympathetic ophthalmia, and uveitis.
- 87. (Currently Amended) The method according to claim <u>80</u> 81, wherein the chronic inflammatory disorder is selected from the group consisting of inflammatory bowel disease <u>is</u> [[,]] Crohn's disease [[,]] <u>or and-ulcerative colitis.</u>
- 88. (Previously Presented) The method according to claim 59, wherein the heterologous protein domain further comprises a human immunoglobulin Fc domain.
- 89. (Currently Amended) The method according to claim 54, wherein the autoimmune disorder is selected from the group consisting of psoriasis, rheumatoid arthritis, diabetes mellitus, multiple sclerosis, sympathetic ophthalmia, and uveitis.
- 90. (Currently Amended) The method according to claim 113 54, wherein the chronic inflammatory disorder is selected from the group consisting of inflammatory bowel disease. 5 is Crohn's disease [[,]] or and ulcerative colitis.

91. (New) The method according to claim 54, wherein the autoimmune disorder is rheumatoid arthritis.

- 92. (New) The method according to claim 70, wherein the parenteral administration is subcutaneous.
- 93. (New) The method according to claim 70, wherein the parenteral administration is intravenous.
- 94. (New) The method according to claim 70, wherein the parenteral administration is intralesional.
- 95. (New) The method according to claim 71, wherein the autoimmune disorder is rheumatoid arthritis.
- 96. (New) The method according to claim 84, wherein the parenteral administration is subcutaneous.
- 97. (New) The method according to claim 84, wherein the parenteral administration is intravenous.
- 98. (New) The method according to claim 84, wherein the parenteral administration is intralesional.
- 99. (New) The method according to claim 81, wherein the autoimmune disorder is rheumatoid arthritis.
- 100. (New) A method for inhibiting lymphotoxin-β-receptor (LT-β-R) signaling in a subject having a Th1 cell-mediated chronic inflammatory disease comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT-β-R blocking agent, and a pharmaceutically acceptable carrier, wherein the LT-β-R blocking agent comprises a soluble LT-β-R.

101. (New) The method according to claim 100, wherein the LT-β-R blocking agent comprises a soluble LT-β-R having a ligand binding domain that can selectively bind to a surface LT ligand.

- 102. (New) The method according to claim 100, wherein the soluble LT-β-R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT-β-R ligand binding domain.
- 103. (New) The method according to claim 100, wherein the LT-β-R blocking agent comprises a soluble LT-β-R fused to one or more heterologous protein domains.
- 104. (New) The method according to claim 103, wherein the heterologous protein domain comprises a human immunoglobulin Fc domain.
- 105. (New) The method according to claim 100, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.
- 106. (New) The method according to claim 100, wherein the pharmaceutical composition is administered to the subject via oral administration.
- 107. (New) The method according to claim 100, wherein the pharmaceutical composition is administered to the subject via parenteral administration.
- 108. (New) The method according to claim 107, wherein the parenteral administration is subcutaneous.
- 109. (New) The method according to claim 107, wherein the parenteral administration is intravenous.
- 110. (New) The method according to claim 107, wherein the parenteral administration is intralesional.

111. **(New)** A method for inhibiting lymphotoxin- β -receptor (LT- β -R) signaling in a subject having a Th1 cell-mediated autoimmune disorder or a Th1 cell-mediated chronic inflammatory disease comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent, and a pharmaceutically acceptable carrier, wherein the LT- β -R blocking agent comprises an antibody directed against LT- β -R.

- 112. **(New)** The method according to claim 111, wherein the autoimmune disorder is selected from the group consisting of psoriasis, diabetes mellitus, multiple sclerosis, sympathetic ophthalmia, rheumatoid arthritis, and uveitis.
- 113. (New) The method according to claim 90, wherein the chronic inflammatory disease is inflammatory bowel disease.